REMARKS

In this Amendment, claim 1 has been amended to incorporate claim 5, and certain limitations of claim 1 are presented as new claim 27. Claims 2 and 5 are canceled without prejudice. Thus, after entry of the Amendment, claims 1, 8 and 27 are all of the claims pending in the application. Applicants submit that no new matter has been entered, and Applicants respectfully request entry of the Amendment.

I. Rejections Under 35 U.S.C. § 112, 1st Paragraph

Written Description

At paragraph 3 of the Office Action, the Examiner rejects claims 1, 2 and 8 under 35 U.S.C. § 112, first paragraph, as allegedly containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the art that the inventors, at the time the application was filed, had possession of the claimed invention. Specifically, the Examiner contends that the specification does not describe any mutant protophorphyringogen oxidase polypeptides, and therefore, claims 1, 2, and 8 encompassing mutant polypeptides do not comply with the written description requirement. The Examiner has not rejected claim 5 under section 112.

Applicants have incorporated claim 5 into independent claim 1. Applicants assert that, since claim 5 has not been rejected under section 112, amended claim 1 satisfies the written description requirement. In addition, Applicants have canceled claim 2, and claim 8 is dependent on claim 1 (via new claim 27). Thus, Applicants assert that amended claims 1 and 8,

and new claim 27 comply with the written description requirement, and Applicants respectfully request withdrawal of this rejection.

Enablement

At page 5 of the Office Action, the Examiner rejects claims 1, 2 and 8 under 35 U.S.C. § 112, first paragraph, because the specification, while being enabling for the isolated polypeptide of SEQ ID NO: 2, allegedly does not provide enablement for mutant protophorphyringogen oxidase polypeptides. The Examiner has not rejected claim 5 under section 112.

Applicants have incorporated claim 5 into independent claim 1. Applicants assert that, since claim 5 has not been rejected under section 112, amended claim 1 satisfies the enablement requirement. In addition, Applicants have canceled claim 2, and claim 8 is dependent on claim 1 (via new claim 27). Thus, Applicants assert that amended claims 1 and 8, and new claim 27 comply with the enablement requirement, and Applicants respectfully request withdrawal of this rejection.

II. Rejections Under 35 U.S.C. § 102(a), (b)

At page 10 of the Office Action, claims 1-2 and 8 are rejected under 35 U.S.C. § 102(a) as anticipated by Lermontova, et al. Specifically, the Examiner contends that claims 1, 2, and 8 encompass the polypeptide disclosed by Lermontova. The Examiner has not rejected claim 5 as anticipated by Lermontova.

Claim 5 has been incorporated into independent claim 1 and claim 2 has been canceled. Accordingly, Applicants assert that Lermontova does not anticipate amended claims 1 and 8, or new claim 27.

In addition, Applicants submit that, while the Examiner contends at page 10 of the office action that *Nt*PPXII maintained protox activity in the presence of 100 nM of acifluorfen, *Nt*PPXII protox activity is not maintained but inhibited (<50% activity) in the presence of 100 nM acifluorfen (see Figure 7 on page 8899, and the last paragraph of the RESULTS section). Lermontova states that, "[t]his level of inhibition indicates that the tobacco mitochondria protoporphyrinogen IX oxidase seems to be more sensitive to acifluorfen than the recombinant enzyme of Arabidopsis...." On the other hand, the Protox of the present invention has a very high tolerance to photobleaching herbicide (see Table 6 of the present specification).

In view of the above, Applicants submit that Lermontova does not teach or suggest amended claims 1 and 8, nor new claim 27. Accordingly, Applicants respectfully request withdrawal of this rejection.

At page 10 of the Office Action, claims 1, 2 and 8 are rejected under 35 U.S.C. § 102(b) as anticipated by Ichinose et al. Specifically, the Examiner contends that claims 1, 2 and 8 encompass the polypeptide disclosed by Ichinose.

Claim 5 has been incorporated into independent claim 1 and claim 2 has been canceled. Accordingly, Applicants assert that Ichinose does not anticipate amended claims 1 and 8, nor new claim 27.

In addition, Applicants submit that Ichinose does not teach or suggest any mutation of protoporphyrinogen oxidase responsible for any photobleaching herbicide resistance, and further, as evident from Watanabe et al. *Biosci Biotechnol Biochem* 2002 Sep;66(9):1799-805 (the

abstract of which is attached herewith), the S23142 tolerance of the YZI-1S cells is due to the over-production of mitochondrial protoporphyrinogen oxidase by gene amplification, and not due to mutation of the gene.

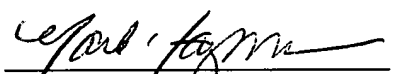
In view of the above, Applicants assert that amended claims 1 and 8, as well as new claim 27, are not anticipated by Ichinose. Accordingly, Applicants respectfully request withdrawal of this rejection.

III. Conclusion

In view of the above, reconsideration and allowance of this application are now believed to be in order, and such actions are hereby solicited. If any points remain in issue which the Examiner feels may be best resolved through a personal or telephone interview, the Examiner is kindly requested to contact the undersigned at the telephone number listed below.

The USPTO is directed and authorized to charge all required fees, except for the Issue Fee and the Publication Fee, to Deposit Account No. 19-4880. Please also credit any overpayments to said Deposit Account.

Respectfully submitted,



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Date: June 18, 2004

Biosci Biotechnol Biochem. 2002 Sep;66(9):1799-805.

Resistance to protoporphyrinogen oxidase-inhibiting compound S23142 from overproduction of mitochondrial protoporphyrinogen oxidase by gene amplification in photomixotrophic tobacco cells.

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Graduate School of Biological Sciences, Nara Institute of Science and Technology, Ikoma, Nara, Japan.

Tobacco YZI-IS cells exhibit a 150-fold greater resistance to the protoporphyrinogen oxidase (Protox)-inhibiting compound, S23142, from wild-type tobacco cells. To investigate the mechanism for this S23142 resistance, the protein level, enzymatic activity, and sensitivity to S23142 in two Protox isoenzymes (plastidal and mitochondrial forms) were examined. The level of mitochondrial Protox protein was greater, and its activity 5-times higher, in YZI-IS cells than in wild-type cells. Furthermore, the apparent IC50 value of S23142 was about 20 nM, which is 20-fold higher than that observed in wild-type cells. In contrast, no differences were found in the plastidal Protox protein level, activity or its inhibition by S23142 between YZI-1S and wild-type cells. A southern blot analysis revealed that the mitochondrial Protox gene had been significantly amplified in the YZI-1S cells. These results suggest that the S23142 resistance of YZI-1S cells was due to the overproduction of mitochondrial Protox by gene amplification.

PMID: 12400676 [PubMed - indexed for MEDLINE]